

Acute, chronic and delayed consequences of noise exposure in animal models – 5 year update

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Noise-induced hearing loss (NIHL) is a serious problem, affecting many millions, worldwide. After acoustic overexposure, hearing thresholds are immediately elevated, but improve with a roughly exponential time course for several weeks, depending on initial severity of the insult and characteristics of the individual. Thresholds may recover fully ('temporary' threshold shift) or may stabilize at an elevated value ('permanent' threshold shift). The acoustic trauma literature suggests that: 1) functionally important structural changes in permanent NIHL involve damage to hair cells and their stereocilia, whereas neural loss after noise is only seen secondarily to loss of inner hair cells; 2) complete threshold recovery indicates reversal of damage to mechanosensory and neural structures and, thus, a benign level of exposure; and 3) delayed changes seen at remote post-exposure times can be attributed to other causes; for example, aging.

New insights into the mechanisms and time course of noise-induced cellular injury and functional loss provided by low-variance mouse models suggest that these basic tenets need re-evaluation. This talk will summarize current understanding of acute and chronic noise-induced hearing loss and cochlear injury, and will discuss how these outcomes are shaped by genetic background, age, and post-exposure time. It will describe recent work documenting delayed neurodegenerative outcomes; progression of inner ear damage long after noise exposure stops, even in ears in which post-exposure threshold sensitivity recovery is complete. Such observations raise important concerns about the long-term effects of apparently benign acoustic overexposures.